## Claims

1. A method for decreasing mitochondrial membrane potential in a mammalian cell, comprising

administering an MHC class II HLA-DR ligand to the mammalian cell to selectively engage MHC class II HLA-DR on the surface of the cell in an amount effective to decrease mitochondrial membrane potential in the mammalian cell, wherein the mammalian cell is not an antigen presenting cell.

2. The method of claim 1, wherein MHC class II HLA-DR is expressed on the surface of the mammalian cell.

3. The method of claim 1, further comprising the step of contacting the mammalian cell with an amount of an MHC class II HLA-DR inducing agent effective to induce the expression of MHC class II HLA-DR on the surface of the mammalian cell.

4. The method of claim 3, wherein the mammalian cell is a tumor cell and wherein the MHC class II HLA-DR ligand is administered to the tumor cell *in vivo* in an amount effective for causing cell lysis of the tumor cell, and wherein the MHC class II HLA-DR inducing agent does not include adriamycin and gamma interferon.

5. The method of claim 3, wherein the MHC class II HLA-DR inducing agent is adriamycin.

6. The method of claim 3, wherein the MHC class II HLA-DR inducing agent is gamma interferon.

7. The method of claim 3, wherein the MHC class II HLA-DR inducing agent is selected from the group consisting of a UCP expression vector, a TCR$\alpha\beta$ engagement molecule and a fatty acid.

8. The method of claim 3, wherein the MHC class II HLA-DR ligand is an anti-

MHC class II HLA-DR antibody.

9. The method of claim 3, wherein the MHC class II HLA-DR ligand is selected from the group consisting of CD4 molecules, αβ T cell receptor molecules, γδ T cell receptor molecules and a MHC class II HLA-DR binding peptide.

10. The method of claim 3, wherein the MHC class II HLA-DR inducing agent and the MHC class II HLA-DR ligand are administered simultaneously.

11. The method of claim 3, wherein the MHC class II HLA-DR inducing agent and the MHC class II HLA-DR ligand are administered orally.

12. The method of claim 3, wherein the MHC class II HLA-DR inducing agent and the MHC class II HLA-DR ligand are administered locally.

13. A method for decreasing mitochondrial membrane potential in a mammalian cell, comprising

contacting the mammalian cell with an amount of an MHC class II HLA-DR inducing agent effective to induce the expression of MHC class II HLA-DR on the surface of the mammalian cell, wherein the mammalian cell is not an antigen presenting cell.

14. A method for increasing mitochondrial membrane potential in a mammalian cell, comprising

administering an MHC class II HLA-DP/DQ ligand to the mammalian cell to selectively engage MHC class II HLA-DP/DQ on the surface of the cell in an amount effective to increase mitochondrial membrane potential in the mammalian cell, wherein the mammalian cell is not an antigen presenting cell.

15. The method of claim 14, wherein MHC class II HLA-DP/DQ is expressed on the surface of the mammalian cell.

16. The method of claim 14, further comprising the step of contacting the

mammalian cell with an amount of an MHC class II HLA-DP/DQ inducing agent effective to induce the expression of MHC class II HLA-DP/DQ on the surface of the mammalian cell.

17. The method of claim 14, wherein the mammalian cell is a pancreatic β cell of a type I diabetic and wherein the MHC class II HLA-DP/DQ ligand is administered to the pancreatic β cell *in vivo*.

18. A method for inducing lysis of a mammalian cell, comprising:

contacting the mammalian cell with an amount of an MHC class II HLA-DR inducing agent effective to induce the expression of MHC class II HLA-DR on the surface of the mammalian cell, and

contacting the MHC class II HLA-DR on the surface of the mammalian cell with an amount of an MHC class II HLA-DR ligand effective for causing lysis of the mammalian cell.

19. The method of claim 18, wherein the MHC class II HLA-DR ligand is an endogenous MHC class II HLA-DR ligand and the step of contacting the mammalian cell with the MHC class II HLA-DR ligand is a passive step.

20. The method of claim 18, wherein the step of contacting the mammalian cell with the MHC class II HLA-DR ligand is an active step.

21. The method of claim 18, wherein the mammalian cell is a tumor cell and wherein the MHC class II HLA-DR ligand is administered to the tumor cell *in vivo* in an amount effective for causing cell lysis of the tumor cell, and wherein the MHC class II HLA-DR inducing agent does not include adriamycin and gamma interferon.

22. The method of claim 18, wherein the mammalian cell is a pancreatic β cell of a type II diabetic and wherein the MHC class II HLA-DR ligand is administered to the pancreatic β cell *in vivo*.

23. The method of claim 18, wherein the MHC class II HLA-DR inducing agent is adriamycin.

24. The method of claim 18, wherein the MHC class II HLA-DR inducing agent is gamma interferon.

25. The method of claim 18, wherein the MHC class II HLA-DR inducing agent is selected from the group consisting of a UCP expression vector, a TCR$\alpha\beta$ engagement molecule and a fatty acid.

26. The method of claim 18, wherein the endogenous MHC class II HLA-DR ligand is an MHC class II HLA-DR expressing cell.

27. The method of claim 18, wherein the MHC class II HLA-DR inducing agent is administered orally.

28. The method of claim 18, wherein the MHC class II HLA-DR inducing agent is administered locally.

29. A method for inducing apoptosis in a tumor cell, comprising:

contacting a tumor cell with an amount of a metabolic modifying agent, which when exposed to a cell causes coupling of electron transport and oxidative phosphorylation, effective to increase the mitochondrial membrane potential in the tumor cell, and

contacting the tumor cell with an amount of an apoptotic chemotherapeutic agent effective for inducing apoptosis in the tumor cell.

30. The method of claim 29, wherein the metabolic modifying agent is glucose.

31. The method of claim 29, wherein the metabolic modifying agent is an MHC class II HLA-DP/DQ ligand.

32. The method of claim 29, wherein the metabolic modifying agent is selected from the group consisting of phorbol myristate acetate in combination with ionomycin, GDP, CD40 binding peptide, sodium acetate, UCP antisense, dominant negative $UCP_{,,}$ and staurosporine.

33. The method of claim 29, wherein the metabolic modifying agent is GDP.

34. The method of claim 29, wherein the apoptotic chemotherapeutic agent is selected from the group consisting of adriamycin, cytarabine, doxorubicin, and methotrexate.

35. The method of claim 29, wherein the metabolic modifying agent and the apoptotic chemotherapeutic agent are administered simultaneously.

36. The method of claim 29, wherein the metabolic modifying agent and the apoptotic chemotherapeutic agent are administered locally.

37. The method of claim 35, wherein the tumor cell is resistant to the apoptotic chemotherapeutic agent.

38. The method of claim 29, wherein the tumor cell is sensitive to the apoptotic chemotherapeutic agent, and wherein the amount of metabolic modifying agent is effective to increase mitochondrial membrane potential and the amount of apoptotic chemotherapeutic agent is effective to inhibit the proliferation of the tumor cell when the mitochondrial membrane potential is increased.

39. A method for decreasing mitochondrial membrane potential in a cell of a subject, comprising

administering an MHC class II HLA-DR ligand to the subject to selectively engage MHC class II HLA-DR on the surface of the cell in an amount effective to decrease mitochondrial membrane potential in the mammalian cell.

40. The method of claim 39, wherein the method is performed in vivo.

41. The method of claim 39, wherein the method is performed ex vivo.

42. The method of claim 39, wherein the mammalian cell is an antigen presenting cell.

43. The method of claim 39, wherein the mammalian cell is selected from the group consisting of a tumor cell and a T cell.

44. A method for inducing the expression of immune recognition molecules on a cell surface, comprising

contacting a cell with an amount of a metabolic inhibition agent effective to decrease mitochondrial membrane potential, wherein a decrease in mitochondrial membrane potential causes induction of the expression of immune recognition molecules on the cell surface.

45. The method of claim 44, wherein the immune recognition molecule is selected from the group consisting of MHC class II, b7-1, b7-2, and CD-40.

46. The method of claim 44, wherein the metabolic inhibition agent is selected from the group consisting of apoptotic chemotherapeutic agents, bacterial byproducts, mycobacterial antigens, UCP expression vectors, and fatty acids.

47. A method for inhibiting pancreatic β cell death in a Type I diabetic, comprising

contacting a pancreatic β cell of a Type I diabetic with an amount of a metabolic modifying agent effective to increase mitochondrial membrane potential in the pancreatic β cell.

48. The method of claim 47, wherein the metabolic modifying agent is selected from the group consisting of glucose, phorbol myristate acetate in combination with

ionomycin, MHC class II HLA-DP/DQ ligand, GDP, and staurosporine.

49. A method for inhibiting pancreatic β cell death in a Type I diabetic, comprising

contacting a pancreatic β cell of a Type I diabetic with an amount of a Fas binding agent effective to inhibit selective engagement of Fas on the surface of the pancreatic β cell.

50. A method for treating a subject having autoimmune disease to reduce associated cell death, comprising

administering an amount of a γδ binding protein effective to specifically bind to and inactivate γδ cells in the subject, wherein the inactivation of the γδ cells inhibits cell death associated with autoimmune disease.

51. The method of claim 50, wherein the γδ binding peptide is an anti-γδ antibody.

52. A method for treating a subject having autoimmune disease to reduce associated cell death, comprising

providing an extracellular environment having a high concentration of glucose to stimulate induction of MHC class II HLA-DP/DQ and a low concentration of fatty acids to inhibit induction of MHC class II HLA-DR, wherein surface expression of MHC class II HLA-DP/DQ is indicative of reduced cell death associated with autoimmune disease.

53. A method for selectively killing a Fas ligand bearing tumor cell, comprising:

contacting the Fas ligand bearing tumor cell with acetate in an amount effective to induce Fas associated cell death.

54. The method of claim 53, wherein the Fas ligand bearing tumor cell is contacted with the acetate in an amount effective to sensitize the cell to a chemotherapeutic agent and further comprising the step of contacting the cell with a chemotherapeutic agent.

55. The method of claim 54, wherein the Fas ligand bearing tumor cell is selected from the group consisting of a melanoma cell and a colon carcinoma cell.

56. The method of claim 53, further comprising the step of administering a Fas ligand to the Fas ligand bearing tumor cell.

57. A method for promoting a Th1 immune response, comprising:

administering to a subject who has been exposed to an antigen an effective amount for inducing a Th1 immune response of a MHC class II HLA-DR inducing agent to induce DR on a T cell.

58. The method of claim 57, wherein the MHC class II HLA-DR inducing agent is fatty acid.

59. A method for screening a tumor cell of a subject for susceptibility to treatment with a chemotherapeutic agent comprising:

isolating a tumor cell from a subject;

exposing the tumor cell to a chemotherapeutic agent; and,

detecting the presence of a cell death marker selected from the group consisting of a Fas molecule on the surface of the tumor cell, a B7 molecule on the surface of the tumor cell, an MHC class II HLA-DR on the surface of the tumor cell, and a mitochondrial membrane potential indicative of cellular coupling wherein the presence of the cell death marker indicates that the cell is susceptible to treatment with a chemotherapeutic agent.

60. The method of claim 59, wherein the cell death marker is a Fas molecule on the surface of the tumor cell and wherein the method comprises the step of contacting the Fas molecule with a detection reagent that selectively binds to the Fas molecule to detect the presence of the Fas molecule.

61. The method of claim 59, wherein the cell death marker is a MHC class II HLA-DR molecule on the surface of the tumor cell and wherein the method comprises the step of contacting the MHC class II HLA-DR molecule with a detection reagent that

selectively binds to the MHC class II HLA-DR molecule to detect the presence of the MHC class II HLA-DR molecule.

62. A method for identifying an anti-tumor drug for killing a tumor cell of a subject comprising:

isolating a tumor cell from a subject;

detecting the presence of a cell death marker selected from the group consisting of a Fas molecule on the surface of the tumor cell, a B7 molecule on the surface of the tumor cell, an MHC class II HLA-DR on the surface of the tumor cell, and a mitochondrial membrane potential indicative of cellular coupling;

exposing the tumor cell to a putative drug; and,

detecting any change in the presence of the cell death marker to determine whether the putative drug is an anti-tumor drug capable of killing the tumor cell of the subject.

63. The method of claim 62, wherein a plurality of tumor cells is isolated from the subject and the plurality of tumor cells is screened with a panel of putative drugs.

64. The method of claim 62, wherein the change in the presence of the cell death marker is detected by contacting the tumor cell with a cell death ligand attached to a solid support.

65. The method of claim 64, wherein the cell death ligand is a Fas ligand.

66. A method for screening a subject for susceptibility to disease, comprising:

isolating a cell selected from the group consisting of peripheral blood lymphocyte and skin from a subject; and,

detecting the presence of an MHC marker selected from the group consisting of an MHC class II HLA-DP/DQ, B7-2, B7-1 and MHC class II HLA-DR on the surface of the cell, wherein the presence of MHC class II HLA-DP/DQ is indicative of susceptibility to atherosclerosis and resistance to autoimmune disease and the presence of MHC class II HLA-DR, B7-2, or B7-1 is indicative of resistance to atherosclerosis and susceptibility to

autoimmune disease.

67. A kit for screening a subject for susceptibility to disease, comprising:

a container housing a first binding compound that selectively binds to a protein selected from the group consisting of B7-2, B7-1 and MHC class II HLA-DR;

a container housing a second binding compound that selectively binds to a MHC class II HLA-DP/DQ protein; and

instructions for determining whether an isolated cell of a subject selectively interacts with the first or second binding compound, wherein the presence of MHC class II HLA-DP/DQ on the cell surface which interacts with the second compound is indicative of susceptibility to atherosclerosis and resistance to autoimmune disease and the presence of MHC class II HLA-DR on the cell surface which interacts with the first compound is indicative of resistance to atherosclerosis and susceptibility to autoimmune disease.

68. A kit for screening a tumor cell of a subject for susceptibility to treatment with a chemotherapeutic agent comprising:

a container housing a cell death marker detection reagent; and

instructions for using the cell death marker detection reagent for detecting the presence of a cell death marker selected from the group consisting of a Fas molecule on the surface of the tumor cell, an MHC class II HLA-DR on the surface of the tumor cell, and a mitochondrial membrane potential indicative of cellular coupling wherein the presence of the cell death marker indicates that the cell is susceptible to treatment with a chemotherapeutic agent.

69. The kit of claim 68, further comprising a container housing a chemotherapeutic agent.

70. The kit of claim 68, further comprising a panel of chemotherapeutic agents, housed in separate compartments.

71. The kit of claim 68, further comprising a cell death ligand.

72. The kit of claim 71, wherein the cell death ligan is coated on a solid surface.

73. The kit of claim 71, wherein the cell death ligand is a Fas ligand.

74. A composition, comprising:
a metabolic modifying agent; and,
an apoptotic chemotherapeutic agent.

75. The composition of claim 74, wherein the metabolic modifying agent is selected from the group consisting of glucose, phorbol myristate acetate in combination with ionomycin, MHC class II HLA-DP/DQ ligand, GDP, and staurosporine.

76. The composition of claim 74, wherein the apoptotic chemotherapeutic agent is selected from the group consisting of adriamycin, cytarabine, doxorubicin, and methotrexate.

77. The composition of claim 74, wherein the metabolic modifying agent and the apoptotic chemotherapeutic agent are present in an amount effective to inhibit the proliferation of a tumor cell.

78. The composition of claim 74, further comprising a pharmaceutically acceptable carrier.

79. A composition, comprising:
an MHC class II HLA-DR inducing agent; and,
an MHC class II HLA-DR ligand.

80. The composition of claim 79, wherein the MHC class II HLA-DR inducing agent is selected from the group consisting of adriamycin, gamma interferon, bacterial byproducts such as lipopolysaccharides, mycobacterial antigens such as BCG, a UCP expression vector, a TCR$\alpha\beta$ engagement molecule and a fatty acid.

81. The composition of claim 79, wherein the MHC class II HLA-DR ligand is selected from the group consisting of CD4 molecules, αβ T cell receptor molecules, γδ T cell receptor molecules and a MHC class II HLA-DR binding peptide.

82. The composition of claim 79, wherein the MHC class II HLA-DR inducing agent and the MHC class II HLA-DR ligand are present in an amount effective to lyse a tumor cell.

83. The composition of claim 79, further comprising a pharmaceutically acceptable carrier.

84. A method for inducing nerve cell differentiation, comprising:

contacting a nerve cell with an amount of a B7 inducing agent effective to induce the expression of B7 on the surface of the nerve cell, and

exposing the nerve cell to a neural activating cell to cause differentiation of the nerve cell.

85. The method of claim 84, wherein the B7 inducing agent is adriamycin.

86. The method of claim 84, wherein the B7 inducing agent is gamma interferon.

87. The method of claim 84, wherein the B7 inducing agent is a fatty acid.

88. The method of claim 84, wherein the B7 inducing agent is a lipoprotein.

89. The method of claim 84, wherein the B7 inducing agent is selected from the group consisting of a B7 expression vector, and a UCP expression vector.

90. The method of claim 84, further comprising the step of contacting the nerve cell with an amount of a metabolic modifying agent, which when exposed to a cell causes coupling of electron transport and oxidative phosphorylation, effective to prevent

dissipation of proton motor force in the nerve cell prior to contacting the nerve with the B7 inducing agent.

91. The method of claim 90, wherein the metabolic modifying agent is glucose.

92. The method of claim 90, wherein the metabolic modifying agent is selected from the group consisting of phorbol myristate acetate in combination with ionomycin, GDP, CD40 binding peptide, sodium acetate, UCP antisense, dominant negative UCP,, and staurosporine.

93. The method of claim 84, wherein the neural activating cell is a T cell.

94. The method of claim 84, wherein the neural activating cell is a macrophage.

95. The method of claim 84, wherein the neural activating cell is a dendritic cell.

96. The method of claim 84, further comprising the step of inducing the expression of a receptor for nerve growth factor.

97. A method for inducing nerve cell differentiation, comprising:

contacting a nerve cell with an amount of a B7 inducing agent effective to induce the expression of B7 on the surface of the nerve cell in the presence of an endogenous neural activating cell.

98. The method of claim 97, wherein the B7 inducing agent is adriamycin.

99. The method of claim 97, wherein the B7 inducing agent is gamma interferon.

100. The method of claim 97, wherein the B7 inducing agent is a fatty acid.

101. The method of claim 97, wherein the B7 inducing agent is the B7 inducing

agent is selected from the group consisting of a B7 expression vector, and a UCP expression vector.

102. The method of claim 97, wherein the B7 inducing agent is a lipoprotein.

103. The method of claim 97, further comprising the step of contacting the nerve cell with an amount of a metabolic modifying agent, which when exposed to a cell causes coupling of electron transport and oxidative phosphorylation, effective to prevent dissipation of proton motor force in the nerve cell prior to contacting the nerve with the B7 inducing agent.

104. The method of claim 103, further comprising the step of administering a fatty acid to the nerve cell to stop cell division.

105. The method of claim 103, wherein the metabolic modifying agent is selected from the group consisting of glucose, phorbol myristate acetate in combination with ionomycin, GDP, CD40 binding peptide, sodium acetate, UCP antisense, dominant negative UCP,, and staurosporine.

106. The method of claim 97, wherein the neural activating cell is a T cell.

107. The method of claim 97, wherein the neural activating cell is a macrophage.

108 The method of claim 97, wherein the neural activating cell is a dendritic cell.

109. A method for inducing apoptosis in a nerve cell, comprising:

contacting a nerve cell with an amount of a metabolic modifying agent, which when exposed to a nerve cell causes coupling of electron transport and oxidative phosphorylation, effective to prevent dissipation of proton motor force in the nerve cell, and

contacting a neural activating cell with an amount of a B7 receptor

blocking agent effective for inducing apoptosis in the nerve cell.

110. The method of claim 109, wherein the metabolic modifying agent is glucose.

111. The method of claim 109, wherein the metabolic modifying agent is selected from the group consisting of phorbol myristate acetate in combination with ionomycin, GDP, CD40 binding peptide, sodium acetate, UCP antisense, dominant negative UCP, and staurosporine.

112. The method of claim 109, wherein the B7 receptor blocking agent is selected from the group consisting of anti-CD28 antibodies, CD28 binding peptides, CTLA4 analogs, anti-CTLA4 antibodies, and CTLA4 binding peptides.

113. A composition, comprising:

a metabolic modifying agent; and,

a B7 receptor blocking agent.

114. The composition of claim 113, wherein the metabolic modifying agent is selected from the group consisting of glucose, phorbol myristate acetate in combination with ionomycin, GDP, and staurosporine.

115. The composition of claim 113, wherein the B7 receptor blocking agent is selected from the group consisting of anti-CD28 antibodies, CD28 binding peptides, CTLA4 analogs, anti-CTLA4 antibodies, and CTLA4 binding peptides.

116. The composition of claim 113, wherein the metabolic modifying agent and the B7 receptor blocking agent are present in an amount effective to induce apoptosis of a nerve cell.

117. The composition of claim 113, further comprising a pharmaceutically acceptable carrier.

118. A composition, comprising:

a B7 inducing agent; and,

a CD28 inducing agent.

119. The composition of claim 118, wherein the B7 inducing agent is selected from the group consisting of adriamycin, gamma interferon, bacterial byproducts such as lipopolysaccharides and lipoproteins, BCG, and fatty acids.

120. The composition of claim 118, wherein the CD28 inducing agent is selected from the group consisting of a T cell receptor engagement molecule, a CD3 engagement molecule, IL4, and a CD28 expression vector.

121. The composition of claim 118, further comprising a pharmaceutically acceptable carrier.

122. A method for re-innervating an injured tissue comprising:

implanting a B7 expressing nerve cell in the injured tissue, wherein the implanted B7 expressing neuron will undergo neuronal differentiation in the presence of a neural activating cell in the injured tissue to re-innervate the injured tissue.

123. The method of claim 122, wherein the B7 expressing nerve cell constitutively expresses B7.

124. The method of claim 123, wherein the B7 expressing nerve cell is a nerve cell which constitutively expresses a UCP gene.

125. The method of claim 123, wherein the B7 expressing nerve cell is a nerve cell which constitutively expresses a B7 gene.

126. The method of claim 122, further comprising administering a B7 inducing agent effective to induce endogenous B7 expression on the surface of the nerve cell.

127. The method of claim 122, wherein the injured tissue is a spinal chord.

128. The method of claim 122, wherein the injured tissue is a severed limb.

129. A method for treating a neurodegenerative disorder, comprising administering an amount of a B7 inducing agent effective to induce the expression of B7 on the surface of a nerve cell.

130. The method of claim 129, wherein the B7 inducing agent is adriamycin.

131. The method of claim 129, wherein the B7 inducing agent is gamma interferon.

132. The method of claim 129, wherein the B7 inducing agent is a fatty acid.

133. The method of claim 129, wherein the B7 inducing agent is an anti-MHC class II HLA-DR antibody.

134. The method of claim 129, wherein the B7 inducing agent is the B7 inducing agent is selected from the group consisting of a B7 expression vector, and a UCP expression vector.

135. The method of claim 129, further comprising the step of inducing expression of CD28 on the surface of a neural activating cell.

136. The method of 135, wherein the neural activating cell is a T cell.

137. The method of claim 135, wherein the neural activating cell is a macrophage.

138. The method of claim 129, wherein the neurodegenerative disorder is selected from the group consisting of Parkinson's disease, Alzheimer's disease,

amyotrophic lateral sclerosis, paralysis, and multiple sclerosis.

139. A method for selectively killing a cell, comprising

contacting the cell with a nucleic acid selected form the group consisting of a UCP anti-sense nucleic acid and a UCP dominant-negative nucleic acid in an amount effect to inhibit UCP function.

140. A method for selectively killing a tumor cell, comprising:

contacting the tumor cell with acetate in an amount effective to induce cell surface Fas expression, and

administering a Fas ligand to the tumor cell in an amount effective to induce Fas associated cell death.

141. The method of claim 140, wherein the tumor cell is contacted with the acetate in an amount effective to sensitize the cell to a chemotherapeutic agent and further comprising the step of contacting the cell with an apoptopic chemotherapeutic agent.

142. A method for selectively killing a tumor cell, comprising:

contacting the tumor cell with a compound selected from the group consisting of acetate and GDP and an apoptopic chemotherapeutic agent in an amount effective to kill the tumor cell.